National Maps of the Effects of Particulate Matter on Mortality: Exploring Geographical Variation

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In this paper, we present national maps of relative rates of mortality associated with short-term exposure to particulate matter < 10 μm in aerodynamic diameter (PM_{10}). We report results for 88 of the largest metropolitan areas in the United States from 1987 to 1994 for all-cause mortality, combined cardiovascular and respiratory deaths, and other causes of mortality. Maximum likelihood estimates of the relative rate of mortality associated with PM_{10} and the degree of statistical uncertainty were obtained for each of the 88 cities by fitting a separate log-linear regression of the daily mortality rate on air pollution level and potential confounders. We obtained Bayesian estimates of the relative rates by fitting a hierarchical model that takes into account spatial correlation among the true city-specific relative rates. We found that daily variations of PM_{10} are positively associated with daily variations of mortality. In particular, the relative rate estimates of cardiovascular and respiratory mortality associated with PM_{10} are larger on average than the relative rate estimates of all-cause and other-cause mortality. The estimated increase in the relative rate of death from cardiovascular and respiratory mortality, all-cause mortality, and other-cause mortality were 0.31% (95% posterior interval, 0.15–0.5), 0.22% (95% posterior interval, 0.1–0.38), and 0.13% (95% posterior interval, –0.05 to 0.29), respectively. Bayesian estimates of the city-specific relative rates ranged from 0.23% to 0.35% for cardiovascular and respiratory mortality, from 0.18% to 0.27% for all causes, and from 0.10% to 0.20% for other causes of mortality. The spatial characterization of effects across cities offers the potential to identify factors that could influence the effect of PM_{10} on health, including particle characteristics, offering insights into mechanisms by which PM_{10} causes adverse health effects. *Key words:* **air pollution, Bayesian methods, hierarchical models, particulate matter, relative rate, spatial smoothing.** *Environ Health Perspect* **111:39–43 (2003). [Online 13 November 2002]**
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Time-series studies conducted in the last decade (*1*,*2*) have shown that air pollution in many cities in the United States, Europe, and other developed countries is associated with increased rates of mortality and morbidity. In interpreting this evidence, scientists have raised concerns about the representativeness of findings from study locations seemingly identified without a sampling plan and about differing modeling strategies among studies. There has also been interest in the variation of effects of particulate matter < 10 μm in aerodynamic diameter (PM_{10}) across the country because particle sources and characteristics are geographically diverse.

The National Morbidity Mortality Air Pollution Study (NMMAPS) (*3*,*4*) was intended to address many of these limitations of evidence derived from time-series studies within single locations and to provide a national-level assessment. The Aerometric Information Retrieval Service (AIRS) database (*5*) maintained by the U.S. Environmental Protection Agency (U.S. EPA) offered a potential sampling frame for selecting study locations based on specific criteria, such as population size and availability of PM_{10} data. Additionally, in 1996 when the NMMAPS began, PM_{10} data for the United States had been collected since 1987, and the monitoring data were sufficiently abundant to support time-series analyses for a number of cities.

A central objective of the NMMAPS was to characterize the effects of PM_{10} and each of the other criteria pollutants alone, and in combination, for the 88 largest U.S. cities. To estimate city-specific, regional, and national air pollution effects, multistage models were developed (*6*,*7*). In the first stage, a separate log-linear regression of the daily mortality rate on air pollution measures and potential confounding factors was fitted to obtain maximum likelihood estimates (MLEs) of the relative rate of mortality associated with the pollution variable and the degree of statistical uncertainty for each city (*8*). In the second stage, the estimates of the relative rates were combined for all cities to obtain an overall estimate and to assess whether city-specific characteristics modify the associations of air pollution and the relative rate of death (*7*).

Previous NMMAPS reports (*3*,*4*) have addressed the estimation of an overall effect by characterizing the heterogeneity of air pollution effects across locations and across geographical regions and have addressed effect modification (*6*,*7*,*9*). For example, Dominici et al. (*7*) investigated whether variability in effect estimates can be characterized by city-specific factors by exploring the dependence of relative mortality rates on mean pollution levels, demographic variables, reliability of the pollution data, and the particle size distribution. Because these analyses were based on data aggregated at the county level, this characterization is limited.

In the present study, we went beyond estimation of a national average pollution effect and created national maps of Bayesian estimates of relative rates of mortality. These were obtained by spatially smoothing the MLEs of relative rates of mortality, taking account of the statistical error in each city's relative rate estimate and the evidence of heterogeneity among the true relative rates. MLEs show more variability in the city-specific estimates than do the maps representing the Bayesian estimates. The MLEs were obtained by using only the time-series data for a particular location. In contrast, the Bayesian estimates were obtained by borrowing strength from neighboring locations; they provide a more sound characterization of the spatial heterogeneity.

The finding of geographical areas with similar city-specific relative rates might indicate city-specific factors that contribute to heterogeneity. For example, we may seek to assess whether the heterogeneity follows broad geographical trends because this might indicate confounding or effect modification by climatic variables. The strengths of the methods used here lie in the synthesis of evidence across broad regions, the quantification of heterogeneity of the effects across cities, and the characterization of the degree of similarity of

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the health effects of air pollution within regions.

We applied a Bayesian hierarchical model that allows for the possible spatial correlation between city-specific estimates to the 88 largest metropolitan areas in the United States from 1987 to 1994, and we report national maps of MLEs and Bayesian estimates of the percentage increase in mortality associated with 10 μg/m^3 increase in PM_{10}. Analyses were conducted for all-cause mortality, cardiovascular and respiratory deaths, and other causes of mortality.

Materials and Methods

We used the NMMAPS database of the largest 90 cities (*3,4*). Because one of the goals of this study was to graphically represent spatial correlation, we excluded Honolulu, Hawaii, and Anchorage, Alaska, from the analysis.

Figure 1 is a map showing the locations of the 88 cities and the 7 geographical regions used in this analysis. The database includes mortality, 24-hr average temperature and dewpoint temperature, and 24-hr average PM_{10} concentration for the 88 largest metropolitan areas in the United States for the 7-year period 1987–1994. The air pollution data were obtained from the AIRS database (*5*) maintained by the U.S. EPA. In some locations, a high percentage of days had missing values for PM_{10} because measurements have been required only every 6 days since 1987 by the agency. These cities were retained, and the resulting increased uncertainty was taken into account in our analysis. We obtained daily cause-specific mortality data, aggregated at the level of the county, from the National Center for Health Statistics (Hyattsville, MD). After excluding deaths from external causes and in nonresidents of the counties, we classified the deaths by age group (< 65, 65–74, and ≥ 75 years) and by cause according to the *International Classification of Diseases*, Ninth Revision: cardiac (codes 390–448); respiratory, including chronic obstructive pulmonary disease and related disorders (codes 490–496), influenza (code 487), and pneumonia (codes 480–486 and 507); and other remaining diseases. The hourly temperature and dew point data for each site were obtained from the Earth Info CD-ROM database (*10*). The database is described in detail elsewhere (*3,4*).

We analyzed the data with a two-stage Bayesian hierarchical model (*11*). At the first stage, we obtained the MLE of the relative rate of mortality associated with a 10 unit change in PM_{10}, $\hat{\beta}^c$, and the corresponding statistical variance v^c within each city, by fitting a log-linear generalized linear model with parametric adjustments for confounding factors. The outcome variable was the total number of deaths on a particular day, the exposure variable was the previous day's PM_{10} level, and the controlled potential confounders were longer term trends, seasonality and weather. No other pollutants were included in the model. The city-specific model specification is similar to the ones used by Kelsall et al. (*8*) and Samet et al. (*3*), but instead of using a generalized additive model with smoothing splines (*12*), we used a generalized linear model with natural cubic splines.

At the second stage, we assumed that the true but unknown city-specific relative rates, β^c, have a common mean, α, and variance, σ^2. We express the degree of similarity of the relative rates in locations c and c' as a function of the distance between the cities. We define the distance between cities as the Euclidean distance of the longitude and latitude coordinates of the cities centroids. More specifically, we assume that $\text{cor}(\beta^c,\beta^{c'}) = \exp\{[-\phi \times d(c, c^c)]^k\}$. The parameter ϕ represents the rate of decay to zero of the correlation as the distance between the two cities increases.

The Bayesian estimate of β, defined as the posterior mean $E[\beta \mid \alpha, \sigma^2, \phi, \text{data}]$, is a weighted average of the MLE, $\hat{\beta}$, and of the overall relative rate, α:

$$E\left[\beta \mid \alpha, \sigma^2, \phi, \text{data}\right] = \Lambda \times \mathbf{1}\alpha + \left(I - \Lambda\right) \times \hat{\beta}, \quad [1]$$

where

$$\Lambda = \left[V^{-1} + \frac{R(\phi)^{-1}}{\sigma^2}\right]^{-1} \times \frac{R(\phi)^{-1}}{\sigma^2} \times \mathbf{1}\alpha\,.$$

Here $\hat{\beta}$ is the vector of the city-specific estimates; V is a diagonal matrix with v^c; $R(\phi)$ is the spatial correlation matrix with off-diagonal elements equal to $\text{cor}(\beta^c, \beta^{c'})$; $\mathbf{1}$ is a vector of ones, and I is the identity matrix. Equation 1 points out that the Bayesian estimate of the city-specific air pollution effects is shrunk toward the overall mean (α), and the shrinkage factor (Λ) is proportional to the statistical uncertainty of the MLEs (V) and to the spatial correlation matrix [$R(\phi)$], but inversely proportional to the degree of heterogeneity of the city-specific relative rates (σ^2).

Model fitting was performed using a Bayesian statistical approach (*13*) and Bayesian software (*14*), which provides an estimate of the posterior distribution of the parameters of interest (α, σ^2, β, ϕ). The posterior distribution was used to determine the probability that the relative rate of mortality associated with PM_{10} has a particular value—that is, it is a measure of the strength of the evidence. A Bayesian estimate is defined as the mean of the posterior distribution. We carried out this analysis without making prior assumptions as to the value of the relative rate. More specifically, prior distributions for β and α were normal with large variances. Prior distribution for σ^{-2} was gamma with scale and shape parameters equal to 0.001 and 0.001. Finally, the prior distribution for ϕ was uniform in the interval (ϕ_{min}, ϕ_{max}). The parameters ϕ_{min} and ϕ_{max} were selected so that if $\phi = \phi_{min}$, the prior correlation at the maximum and at the minimum distance was 0.01–0.82, and if $\phi = \phi_{max}$, the prior correlation at the maximum and at the minimum distance was 0–0.52.

We used the posterior distribution to determine the probability that the relative rate of mortality associated with PM_{10} is in a particular interval; it can also be used to determine the 95% posterior intervals. The 95% posterior interval encompasses 95% of the posterior distribution, a Bayesian formulation analogous to the 95% confidence interval. To approximate the posterior distributions of all the parameters of interest, we implemented simulation-based methods, and in particular the Geobugs software (*14*). Statistical models for analyzing correlated geographic cohort data based on Cox proportional hazards survival model with spatial correlated random effects have been proposed by Burnett et al. (*15*).

Results

Figure 2 shows the posterior distributions of the overall effects (α) and of the spatial correlation, $\text{cor}(\beta^c, \beta^{c'})$, for total mortality, cardiovascular–respiratory mortality, and other-cause mortality. We found that the estimated overall relative rate of cardiovascular–respiratory mortality associated with PM_{10} (percent increase in mortality per 10-μg/m^3 increase in PM_{10}) was the highest (0.31%; 95% posterior interval, 0.15–0.5) compared to estimated overall relative rates of death for total mortality and other causes of mortality at 0.22% (95% posterior interval; 0.1–0.38), and 0.13% (95% posterior interval; –0.05 to 0.29), respectively.

Between-city standard deviation indicates the degree of heterogeneity of the relative rates for mortality across cities with respect to the overall relative rate. For example, if the overall relative rate, α, equals 0.22 and the between-city standard deviation, σ, equals

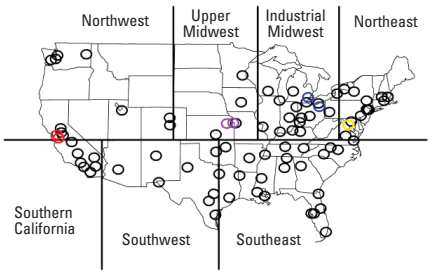

Figure 1. Locations of the 88 largest U.S. cities with the seven geographical regions. The city pairs corresponding to the four distances shown in Table 1 are indicated by yellow (Washington, DC, and Arlington, VA), red (San Jose, CA, and Oakland, CA), purple (Kansas, KS, and Topeka, KS), and blue (Detroit, MI, and Akron, OH) circles.

0.07 (as for total mortality), then the true city-specific relative rates are within the interval (0.22 ± 1.96 × 0.07) with an approximate probability of 95%. The relative rates of total mortality, cardiovascular and respiratory mortality, and other causes of mortality have similar degrees of heterogeneity with posterior means of σ equal to 0.07 (95% posterior interval; 0.02–0.33), 0.08 (95% posterior interval; 0.02–0.50), and 0.08 (95% posterior interval; 0.02–0.34), respectively.

Figure 2B shows the posterior distributions of the spatial correlation cor(β^c, $\beta^{c'}$) = exp (–ϕ × distance between c and c') at the four distances 0.08, 0.48, 1, and 2 for total mortality. The spatial correlation indicates the degrees of smoothness of the relative rates of mortality for one city with respect to the neighboring cities. The city pairs corresponding to the four distances are indicated in Figure 1. As expected, the degree of spatial smoothness is largest for the closest cities (Arlington, VA, and Washington, DC) with a 95% posterior interval for the spatial correlation varying from 0.52 to 0.97. Cities with distances of 2 (Detroit, MI, and Akron, OH) have much lower spatial correlation with 95% posterior intervals ranging from 0.04 to 0.35 (Table 1). Cities with distance > 2 have a posterior distribution of the spatial correlation coefficient concentrated at zero.

Figures 3 and 4 show MLEs and Bayesian estimates of the city-specific relative rates for total, cardiovascular–respiratory, and other causes of mortality. The MLEs are obtained by using only that city's data. The Bayesian estimates are obtained by spatially smoothing the crude estimates with a degree of smoothness estimated from the data.

In Figures 3 and 4, the areas of the circles are proportional to the precisions (inverse of the variances) of the MLEs and Bayesian estimates, with larger circles indicating more precise estimates. The precision of the MLE depends on the number of days that PM_{10} was recorded and on the number of mortality events. Relative rates with t-ratio > 1.96 are indicated in Figures 3 and 4. In the Bayesian maps (Figure 4), the t-ratios are approximated by the ratios between the posterior means and the posterior standard deviations of the city-specific relative rates.

The city-specific MLEs (Figure 3) for all outcomes combined vary from –4 to 4% increase in mortality per 10 μg/m^3 increase in PM_{10}. Their spatial variation is shown by a continuous color scale ranging from blue, yellow, red, and purple (Figure 3).

The city-specific Bayesian estimates (Figure 4) are heavily shrunk toward their overall mean. For all outcomes combined, the Bayesian estimates vary from 0.1 to 0.35% increase in mortality per 10 μg/m^3 increase in PM_{10}. Figure 4 shows spatial variation with a continuous color scale ranging from yellow, red, and purple, but with different cutoffs than in the MLE maps.

Both the MLE maps (Figure 3) and the Bayesian maps (Figure 4) indicate that relative rates of total, cardiovascular–respiratory diseases, and other causes of mortality are larger in the Northeast and in the southern California regions. In addition, shrinkage and spatial smoothing increase the precision of the Bayesian estimates as shown by a larger number of circles with black outlines in the Bayesian maps (Figure 4) than in the MLE maps (Figure 3).

Discussion

Particulate air pollution is a national public health problem, regulated under the provisions of the Federal Clean Air Act. Using national data, we attempted to characterize the effect of particulate air pollution on mortality for the largest cities in the United States. We used Bayesian methods to map the relative mortality rates associated with PM_{10}, grouping the nation into seven regions, following the regions designated by the U.S. EPA.

We found that there was some modest variation in the relative risks across the nation (Figures 3 and 4). In previously reported analyses, we were unable to explain the heterogeneity using descriptors of the population, air pollution characteristics, and reliability of the PM_{10} measurement data (*8*).

Beyond random variation alone, the heterogeneity has several potential and nonexclusive explanations: across-region variation in the characteristics and sizes of the populations susceptible to air pollution and variation in the toxicity of PM_{10}. With regard to susceptibility, persons with underlying heart and lung disease, particularly the elderly, have been postulated to be at increased risk from exposure to PM_{10} or other air pollutants (*16*). Both children and adults with asthma may also be at increased risk. Variation in the frequency of chronic heart and lung disease across the country is well documented. Mortality rates from chronic obstructive pulmonary disease and coronary heart disease vary widely, being highest in the Southeast and lowest across the mountain West (*17*). The range of age-adjusted mortality rates is approximately 2-fold, indicating an approximately similar range in prevalence. Asthma rates also vary, tending to be higher in inner cities with high proportions of minority children (*18,19*). Correspondence has not been found between indicators of the relative sizes of susceptible populations across the country and maps of comparative pollution effects.

Sources of airborne particulate matter vary across the country, as does the chemical composition and size distribution of particulate matter (*20,21*). Nationally, primary particulate emissions come from fugitive dust, biomass burning, agriculture, wind erosion, fossil-fuel combustion, and other sources; secondary particles are formed from the precursor gases

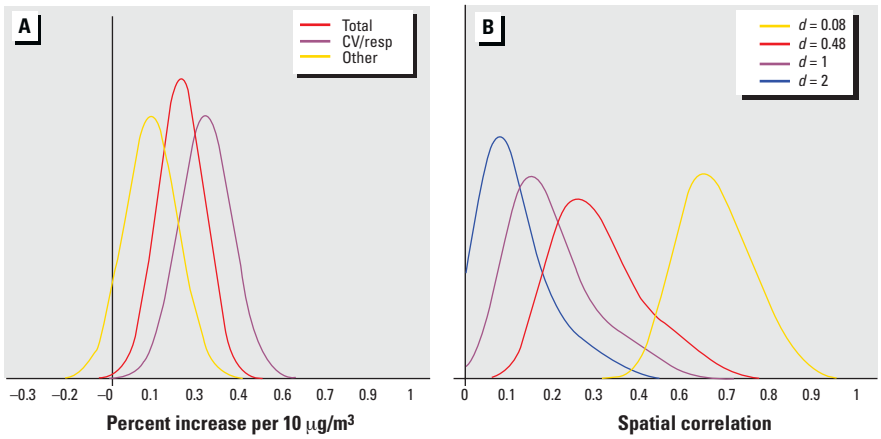

Figure 2. (*A*) Marginal posterior distributions of the overall effects, α, for total mortality (Total), cardiovascular–respiratory mortality (CV/resp), and other causes of mortality (Other), based on the percent increase in mortality per 10-μg/m^3 increase in PM_{10}. The curves indicate the posterior means. (*B*) Marginal posterior distributions of the spatial correlation, cor(β^c, $\beta^{c'}$), evaluated at four distances (*d*).

Table 1. Posterior means and posterior quantiles of the spatial correlation cor(β^c,$\beta^{c'}$) = exp {–[ϕ × distance (c,c')]$^{0.5}$} for selected distances.

		Quantile		
Distance	Cities	2.5%	50%	97.5%
0.08	Washington, DC, and Arlington, VA	0.52	0.63	0.97
0.48	San Jose, CA, and Oakland, CA	0.20	0.32	0.59
1.00	Kansas, KS, and Topeka, KS	0.10	0.19	0.47
2.00	Detroit, MI, and Akron, OH	0.04	0.09	0.35

Examples of city-pairs at the four distances are indicated in yellow (0.8), red (0.48), purple (1), and blue (2) in Figure 1.

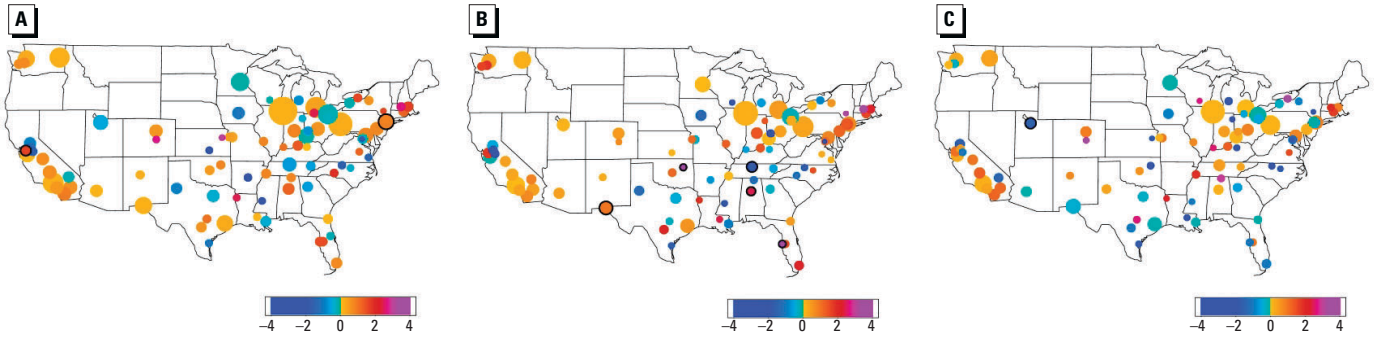

Figure 3. MLEs of the log relative rates of mortality from exposure to PM_{10} (percent increase in mortality per 10-μg/m^3 increase in PM_{10}) for (*A*) total mortality, (*B*) cardiovascular–respiratory mortality, and (*C*) other causes of mortality. The areas of the circles are proportional to the statistical precisions of the MLEs; larger circles indicate more precise estimates. City-specific MLEs are calculated separately within each city. The circles with the black outline denote the relative rates that are statistically different from zero. The city-specific MLEs for all outcomes combined vary from –4 to 4% increase in mortality. MLEs are color coded as follows: blue, < 0; yellow, 0–1; red, 2–3; purple, > 4.

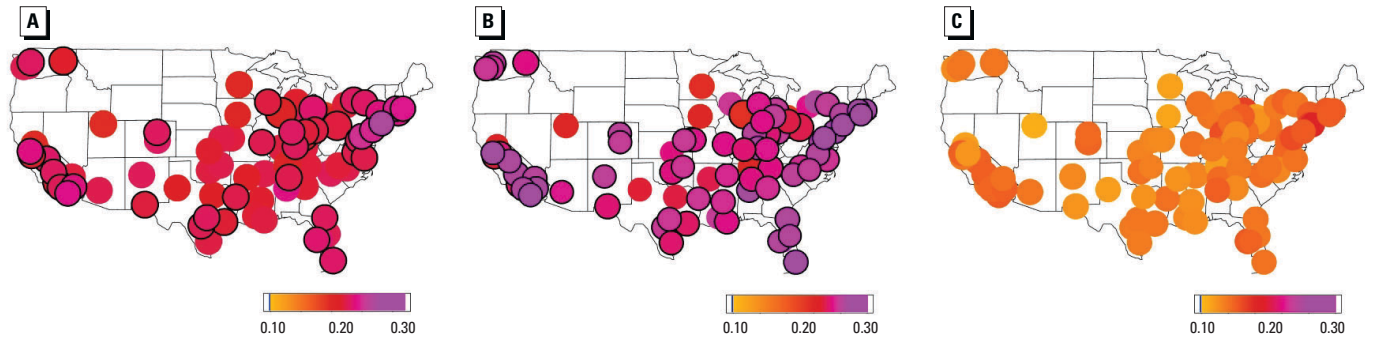

Figure 4. Bayesian estimates of the log relative rates of mortality from exposure to PM_{10} (percent increase in mortality per 10-μg/m^3 increase in PM_{10}) for (*A*) total mortality, (*B*) cardiovascular–respiratory mortality, and (*C*) other causes of mortality. The areas of the circles are proportional to the posterior precisions of the Bayesian estimates; larger circles indicate more precise estimates. City-specific Bayesian estimates are spatially smoothed with respect to neighboring cities and with respect to their overall mean. The circles with the black outline denote relative rates with posterior mean and posterior standard deviation ratio > 1.96. For all outcomes combined, the Bayesian estimates vary from 0.1 to 0.35% increase in mortality. Bayesian estimates are color coded as follows: yellow, 0.1–0.2; red, 0.2–0.25; purple, > 0.35. Note that there is no correspondence between the color scale in this figure and the color scale in Figure 3.

sulfur dioxide and nitrogen dioxide and volatile organic compounds.

Some general conclusions can be made about regional differences in particle composition (*20,21*). In the eastern United States, secondary particles appear to dominate particulate matter ≤ 2.5 μm in aerodynamic diameter ($PM_{2.5}$), whereas crystal dusts are prominent in agricultural areas and in desert regions. Comparative data for the eastern and western United States show that $PM_{2.5}$ particles have a greater proportion of sulfate and less organic carbon in the eastern portion of the country. We cannot yet, however, link specific particle characteristics to toxicity (*22*); this topic is a focus of intense research as recommended by the National Research Council's Committee on Research Priorities for Airborne Particulate Matter (*23*). Concentrations of PM_{10} and $PM_{2.5}$ vary across the country, as does their ratio. If, in fact, the smaller particles are the component of airborne particulate matter causing increased mortality, we would anticipate the greatest effects in those regions having the highest concentrations of $PM_{2.5}$, regardless of PM_{10} concentration. The 1999 data from the U.S. EPA, although still incomplete, indicate the highest levels in California and across the Midwest and Southeast. This pattern is only partially concordant with the mortality maps in Southern California and in the Midwest, but not concordant with the mortality pattern found in the Northeast.

We have also found that the effect of PM_{10} on mortality is negatively modified by the PM_{10} level itself; that is, the effect of PM_{10} per unit concentration declines at increasing concentrations (*4,7*). The maps of risks associated with PM_{10} (Figures 3 and 4) are not consistent with this pattern of modification. With further characterization of particles across the country from new monitoring initiatives, a richer database will be available to explore variation in health risk in relation to the heterogeneity of particle characteristics.

Our mapping strategy represents a starting point for refinement. In our modeling strategy, we assumed a similarity of the relative rates within regions based on the Euclidean distance between the cities. This simplistic assumption was necessitated by a lack of additional, external information on factors that might drive heterogeneity of risk estimates. The modeling approach might be refined by incorporating relevant geographic and meteorologic characteristics as well. One enhancement would be to incorporate priors based on results of receptor models, which would integrate sources and meteorology to provide more credible priors. The finding of heterogeneity has potential implications with regard to research opportunities and public health protection. The heterogeneity in risk estimates offers an opportunity to perform hypothesis-driven research, assessing the consistency of hypotheses concerning toxicity of particles against the observed differences in risk. At present, the National Ambient Air Quality Standards (*24*) are set on mass alone. A more complete understanding of the causes of heterogeneity of risk might lead to more focused source control or even to standards directed at specific types of particles.

References and Notes

1. Dockery D, Pope CA, Xu X, Spengler J, Ware J, Fay M, Ferris B, Speizer F. An association between air pollution and mortality in six U.S. cities. N Engl J Med 329:1753–1759 (1993).

2. Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society. Health effects of outdoor air pollution, Part 1. Am J Respir Crit Care Med 153:3–50 (1996).
3. Samet JM, Zeger SL, Dominici F, Dockery D, Schwartz J. The National Morbidity, Mortality, and Air Pollution Study (HEI Project No. 96-7): Methods and Methodological Issues. Cambridge, MA:Health Effects Institute, 2000.
4. Samet JM, Zeger SL, Dominici, F, Curriero F, Coursac I, Dockery D, Schwartz J, Zanobetti A. The National Morbidity, Mortality, and Air Pollution Study (HEI Project No. 96-7): Morbidity and Mortality from Air Pollution in the United States. Cambridge, MA:Health Effects Institute, 2000.
5. Aerometric Information Retrieval Service (AIRS) Database. Available: http://www.epa.gov/air/data/info.html [cited 11 October 2002].
6. Dominici F, Samet JM, Zeger SL. Combining evidence on air pollution and daily mortality from the twenty largest US cities: a hierarchical modeling strategy. R Stat Soc Ser A 163:263–302 (2000).
7. Dominici F, Daniels M, Zeger SL, Samet JM. Air pollution and mortality: estimating regional and national dose-response relationships. J Am Stat Assoc 97:100–111 (2002).
8. Kelsall J, Samet JM, Zeger SL. Air pollution, and mortality in Philadelphia, 1974–1988. Am J Epidemiol 146:750–762 (1997).
9. Samet JM, Dominici F, Curriero F, Coursac I, Zeger SL. Fine particulate air pollution and mortality in 20 U.S. cities: 1987-1994. N Engl J Med 343(24):1742–1757 (2000).
10. Earth Info CD-ROM Database. Available: http://www.earthinfo.com/databases/databases.htm [cited 11 October 2002].
11. Lindley DV, Smith AFM. Bayes estimates for the linear model. J R Stat Soc Ser B Methodologic 34:1–41 (1972).
12. Hastie TJ, Tibshirani RJ. Generalized Additive Models. New York: Chapman and Hall, 1990.
13. Gelman A, Rubin DB. Inference from iterative simulation using multiple sequences. Stat Sci 7:457–472 (1992).
14. Best N. Geobugs. Available: http://www.mrc-bsu.cam.ac.uk/bugs/winbugs/geobugs.shtml [cited 11 October 2002].
15. Burnett R, Ma R, Jerrett M, Goldberg MS, Cakmak S, Pope CA III, Krewski D. The spatial association between community air pollution and mortality: a new method for analyzing correlated geographic cohort data. Environ Health Perspect 109(suppl 3):375–380 (2001).
16. American Thoracic Society. What constitutes an adverse health effect of air pollution? Official statement of the American Thoracic Society. Am J Respir Crit Care Med 161:665–673 (2000).
17. Pickle LW, Mungiole M, Jones GK, White A. Atlas of United States Mortality. Hyattsville, MD:National Center for Health Statistics, 1996.
18. Centers for Disease Control and Prevention. Asthma mortality and hospitalization among children and young adults– United States, 1980-1993. MMWR Morb Mortal Wkly Rep 146:177–183 (1996).
19. Weiss KB, Wagener DK. Changing patterns of asthma mortality. Identifying target populations at high risk. JAMA 264:1683–1687 (1990).
20. U.S. EPA. Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information. OAQPS Staff Paper. Research Triangle Park, NC:U.S. Environmental Protection Agency, 1996.
21. U.S. EPA, Office of Research and Development. Air Quality Criteria for Particulate Matter: Second External Review Draft. Washington DC:U.S. Environmental Protection Agency, 2001.
22. National Research Council. Research Priorities for Airborne Particulate Matter. I. Early Research Progress. Washington, DC:National Academy Press, 1998.
23. National Research Council. Research Priorities for Airborne Particulate Matter III. Early Research Progress. Washington, DC:National Academy Press, 2000.
24. U.S. Environmental Protections Agency. National Ambient Air Quality Standards (NAAQS). Available: http://www.epa.gov/airs/criteria.html [cited 11 October 2002].